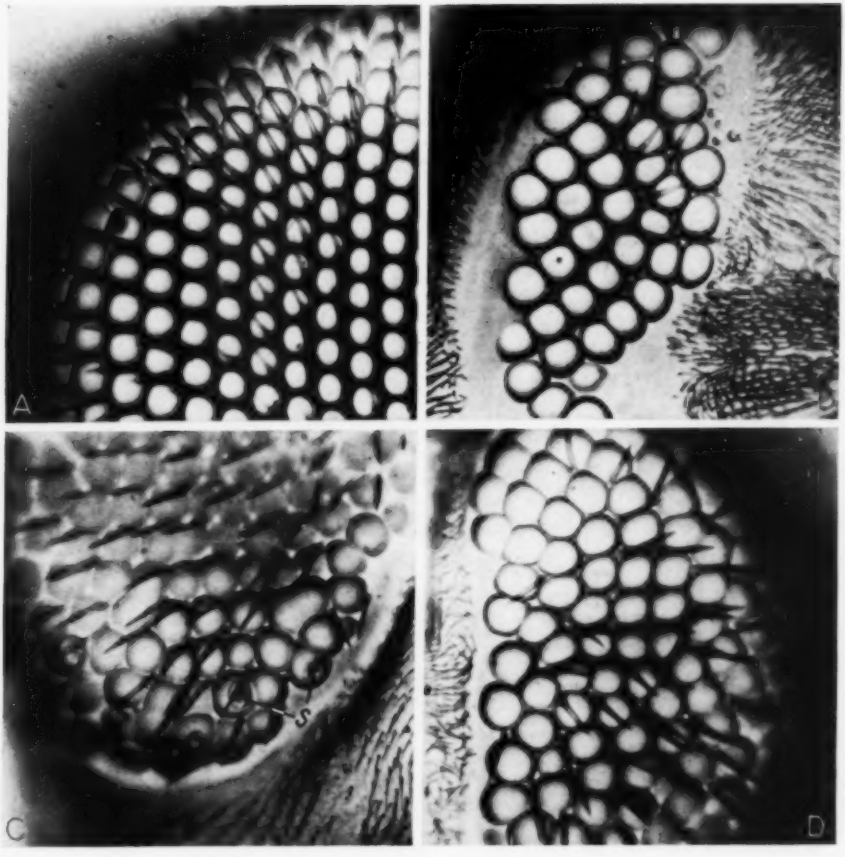
PEARL

The Journal of HEREDITY



GENES AND THE DROSOPHILAN EYE

(SEE PAGE 395)

The Naked Fowl

The Midget Tomato

Artificial Insemination in Russia

The "Kindred File" in Human Genetics

VOLUME 29 OCTOBER, 1938 NUMBER 10

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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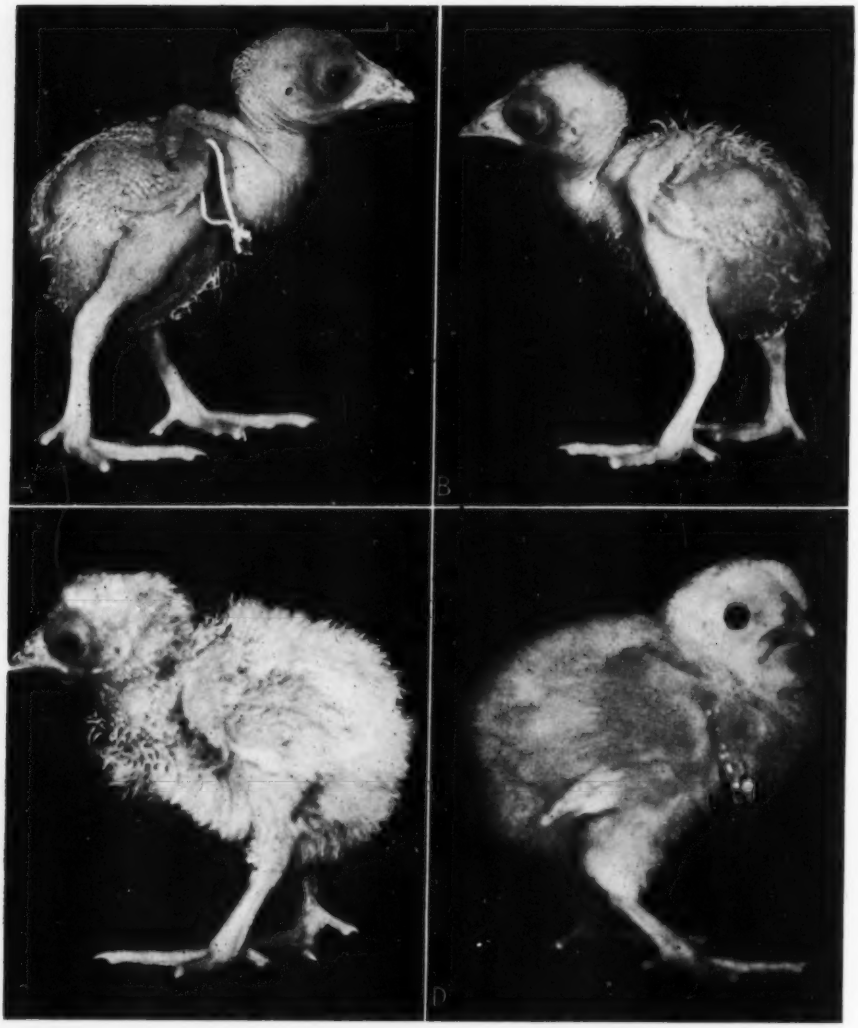


NAKED AND NORMAL CHICKS

Frontispiece

Newly hatched chicks showing three degrees of nakedness are compared with a normal chick (*D*). All of the three "naked" chicks (*A*-*C*) carry the *n* gene, and they show the range of variation observed. Chick *C* approaches normality more closely than any other of 150 naked chicks thus far hatched. The majority are like *A* and *B*.

GENETICS OF THE FOWL.

IX. Naked, A New Sex-Linked Mutation

F. B. Hutt and P. D. Sturkie

Department of Poultry Husbandry, Cornell University, Ithaca, New York

A WHITE Leghorn chick closely resembling that shown in the Frontispiece (*A*) was brought in to this laboratory in 1935. The owner, who sought an explanation of the abnormality, stated that during the previous three years several others had occurred in the flock from whence it came. Fortunately, the chick lived to maturity. Genetic studies of its descendents have shown that the loss of down is caused by a recessive sex-linked mutation which is lethal to about half of the affected chicks during the last two or three days of incubation.

Description

A. Chicks

At hatching affected chicks show degrees of feathering varying from an almost complete lack of down to the condition shown in Frontispiece-*C* which shows the most completely covered chick yet obtained among over 150 naked chicks hatched from this stock. The majority are like the naked specimen shown in Frontispiece-*A* or the somewhat intermediate chick (*B*).

In the sparsely covered chicks there is complete absence of down in the apteria and the few strands of down on the pterylae seem wiry, short, thickened and sometimes curled or twisted. This appearance is caused by failure of the barbs to separate as in the normal feather (Figure 1). So firmly are they stuck together that even after soaking in water or alcohol it is difficult to separate them. In the modified naked chick (Frontispiece *B* and *C*) the down feathers show varying degrees of approach to the normal condition and some of them on the posterior spinal pteryla and in the abdominal region are indistinguishable from normal down feathers.

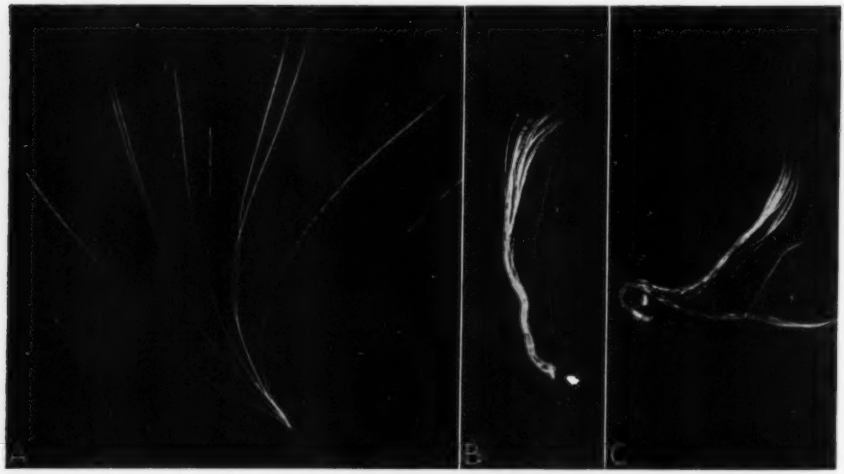
In general the anterior end of the chicks is more naked than the posterior one. The head, anterior spinal and alar feather tracts, as well as the anterior part of the ventral tract, may be quite bare, while the others are comparatively well covered, particularly the posterior spinal tract. This tendency toward an antero-posterior gradient of decreasing effect of the mutation is partially evident in the Frontispiece.

The arrangement and extent of the feather tracts are normal, and the follicles are quite distinct, somewhat as in a chick from which the down has been plucked.

Naked and normal chicks do not differ significantly in size at hatching, but the latter appear much larger because of their fluffy down (Frontispiece-*D*).

B. Juveniles

From about two to four weeks of age most of the naked chicks appear more naked than at any other period of their lives. The sparse, wiry down is worn off and feathers replacing it are very slow to develop. Even chicks partially covered at hatching may be quite naked in appearance at three weeks. In comparison with normal birds, this effect is heightened by the almost complete failure of the juvenile remiges to develop (Figure 2). The head tract is slowest in feathering and the humeral and femoral tracts usually show the longest feathers. The juvenile primary feathers do not appear in regular sequence from the inner to the outer one as in normal chicks. Some chicks may have at four weeks only the inner primary and one near the tip of the wing, perhaps the seventh or eighth, broken through the skin. Feather follicles not erupted fre-



BARBS OF "NAKED" FEATHERS DEFECTIVE

Figure 1

Single chick down feathers from normal (*A*) and naked (*B, C*) chicks. In the latter the barbs are folded and twisted together to form a single strand which, when softened, can be separated into its constituent barbs (*C*). This reveals that barbules are present in the abnormal feather as in the normal one.

quently appear swollen as if some constriction were preventing their contents from being pushed out. The rectrices are more successful than the remiges in breaking through their sheaths. In both a central rachis is present but the barbs are frequently spread out to form a rather downy feather like that of the Silky. Many of these feathers appear to be broken off right at the follicle, others at one to three centimeters from the skin.

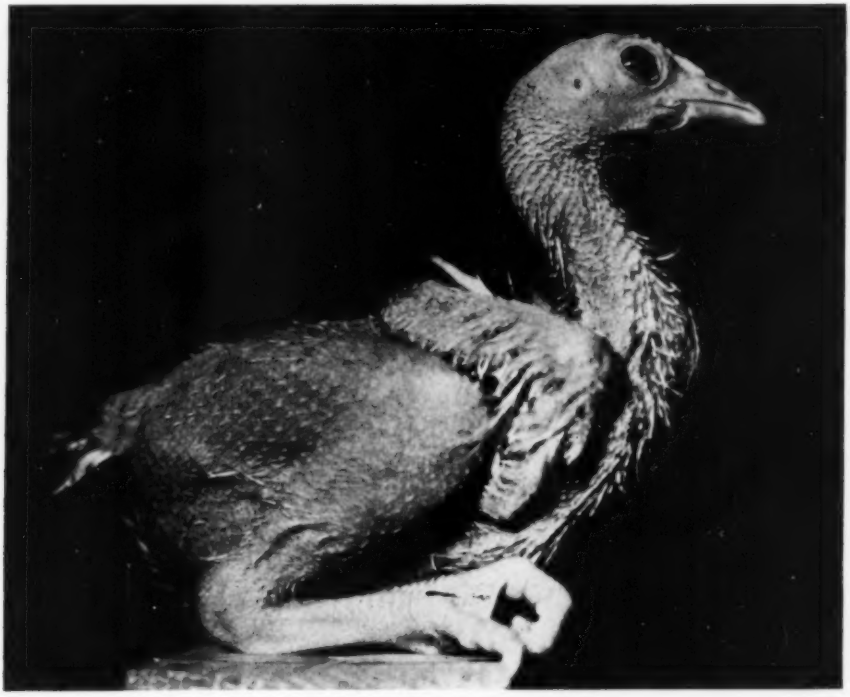
C. Adults

Mature birds have a much more complete covering of feathers than young ones but there is a great deal of variability in the extent of feathering. Pterylosis is normal. There are feathers on all of the pterylae, and down feathers in the apteria. The fluff plumage of the abdominal and ventral regions may be almost normal in density and length of feather (Figure 3*A*). Elsewhere the tips of the feathers are usually broken off and some have unusually long barbs at the distal end (Figure 4), so that the birds have a rough plumage instead of a smooth one.

The remiges are usually all missing, but stubs may remain of one or two that have broken off. The rectrices are better represented and up to four of them have been counted. Some of these attain full size and appear normal.

Areas lacking plumage usually show stubs of feathers which have broken off at distances of about one centimeter or less from the skin (Figure 3*B*). Some seem broken right inside the follicle. Some have blood clots at the broken end, indicating that the break has come shortly after eruption of the feather from the follicle sheath. In others the follicle is filled with dried blood. Subcutaneous hemorrhages are found in some follicles from which the feathers have not erupted. The scales of the feet are normal.

Under the microscope feathers from mature naked fowls are seen to be normal except that in most of them the apical barbs and the tip of the rachis are broken off (Figure 4). Some of the barbs are unusually long and lack



MAXIMUM NAKEDNESS

Figure 2

A naked chicken at five weeks of age, at about the end of the period when the feathers are most defective. The distribution of feather follicles is normal but the feathers are few and short.

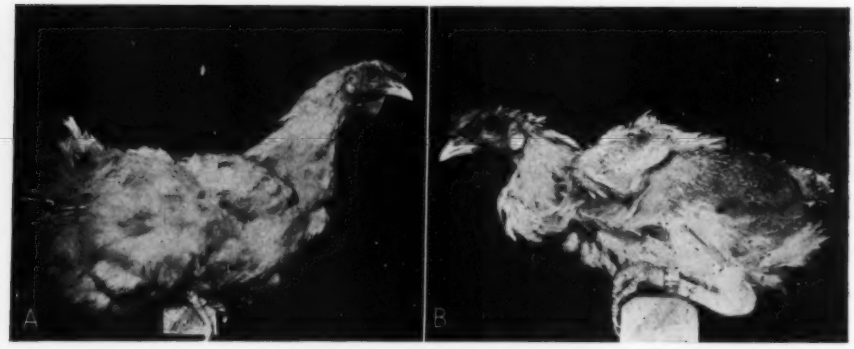
barbules on a greater portion of their distal extremities than do those on normal feathers. This gives the feathers of females somewhat the appearance of typical male neck and wing feathers, except that the feather is more rounded and the tip is broken off. Elsewhere, where barbules are present, they carry hamuli and barbicels like those in normal feathers and the result is a normal web.

Genetics

The original naked White Leghorn chick, secured in 1935, turned out to be a female. In the spring of 1936 she was mated (by artificial insemination) with an unrelated normal White Leghorn male and produced 13 chicks, all with normal down. Unfortunately, the original naked hen did not survive for another year and a backcross to her in 1937 was impossible. However, in that year one of her sons was mated to four of his full sisters and to some unrelated hens. Another son was mated with 12 Rhode Island Red hens.

It was soon apparent that both of these F_1 males produced featherless chicks just as readily from unrelated hens as from full sisters, that all of the naked ones (in these crosses) were females, and that considerably more than half of the affected embryos died during the last three days of incubation.

Four more such matings — of heterozygous males to females not carrying



MATURE NAKED FEMALES

Figure 3

The one on the left has almost normal plumage in the ventral regions and on the abdomen, but lacks the large wing feathers entirely. This is the extreme variation toward normality. The one on the right has only a scanty covering, and lacks both wing and tail feathers. The original naked chicken had fewer feathers than that in (*B*), but plumage in most adults is between the extremes shown.

the gene — were made in 1938. The results in all six of these matings are tabulated in Table I.

Assuming that there is no differential mortality of normal and naked chicks prior to the last three days of incubation, the expectation for a sex-linked recessive mutation is that among those hatched or dying during the last three days of incubation, one-quarter would be featherless. There is a close fit of observed to expected ratios in each of the six matings reported in Table I, and altogether there are 712 normal : 246 naked where the theoretical expectation is 719:239.

Evidence of the six-linked nature of the mutation is shown in Table I and summarized (for the three matings in which sexes of all hatched chicks are known) by the following sex-ratios among 477 chicks hatched, or dead at 19-21 days of incubation:

	Normal		Naked	
	♂♂	♀♀	♂♂	♀♀
Observed	239	104	0	134
Expected	239	119	0	119

The observed numbers fit quite well to the expectation that among chicks not naked there should be a ratio of 2♂♂ : 1♀, and no males whatever among the naked chicks.

It is proposed to call this mutation "naked" and to designate it by the symbol *n*, which, so far as the writers can ascertain, has not yet been preempted for any other mutation in the fowl.

No effects of the gene are noticeable in heterozygous males. During the past six months matings were made to determine the effect of the gene on homozygotes. One might reasonably have expected that a mutation having such a marked effect in hemizygotes, and lethal to more than half of them, would have still greater effects upon the homozygous males. Such, however, is not the case.

Table I. Classifications in genetic analysis of the naked stock.

Male	Dead at 19-21 days of incubation		Hatched		Ratios Normal:Naked	
	Normal	Naked	Normal	Naked	Observed	Expected †
	♂♂ ♀♀	♂♂ ♀♀	♂♂ ♀♀	♂♂ ♀♀	♂ n	♂ n
Matings of Rh ♂♂ x N- ♀♀						
C 5919	17 7	0 39	75 37	0 81	136: 60	147: 49
C 5913	4 2	0 24	65 15	0 5	90: 29	85: 30
D 5976 (a)	6 2	0 21	72 37	0 26	117: 45	121: 40
D 5976 (b)	7 4	0 9	78*	28*	89: 30	89: 30
D 5983	5 3	0 19	180*	57*	186: 56	183: 64
D 5983	0 1	0 3	91*	27*	91: 26	89: 29
				Total	712:246	719:239
Matings of Rh ♂♂ x n- ♀♀						
D 5977	3 0	2 6	14 13	7 8	30: 25	27.5: 27.5
D 5980	1 2	5 4	3 7	2 5	13: 16	14.5: 14.5
Total	4 2	7 10	17 20	9 13	43: 41	42.0: 42.0

* These birds could not be classified for sex at the time of writing, but of the 41 naked chicks, 47 which have died were all females.

† To the nearest whole number.

Two males proven heterozygous were mated with sibling naked females. To avoid injuries during treading to the partially naked backs of these females, one mating was made by artificial insemination (♂ D 5977) and for the other, in which the hens were somewhat better covered, the male was placed in the pen only on every third day. The results, shown in the lower part of Table I, indicate that homozygous males suffer no higher mortality than hemizygous females during the last three days of incubation. It would also appear, since the ratio of 43 normal : 41 naked among those that hatched or died after 18 days of incubation is the same as the 1 : 1 zygotic ratio, that none of the naked chicks in these matings died earlier than 19 days. However, among the 41 naked there were 16♂♂ : 25♀♀. This is probable just a chance deviation from the 20.5 expected for each sex, but, if further data in matings of this type continue to show an excess of females, they would indicate that the mutation is lethal to some of the homozygotes at an earlier age than to the hemizygotes.

Lethal Action of the Gene

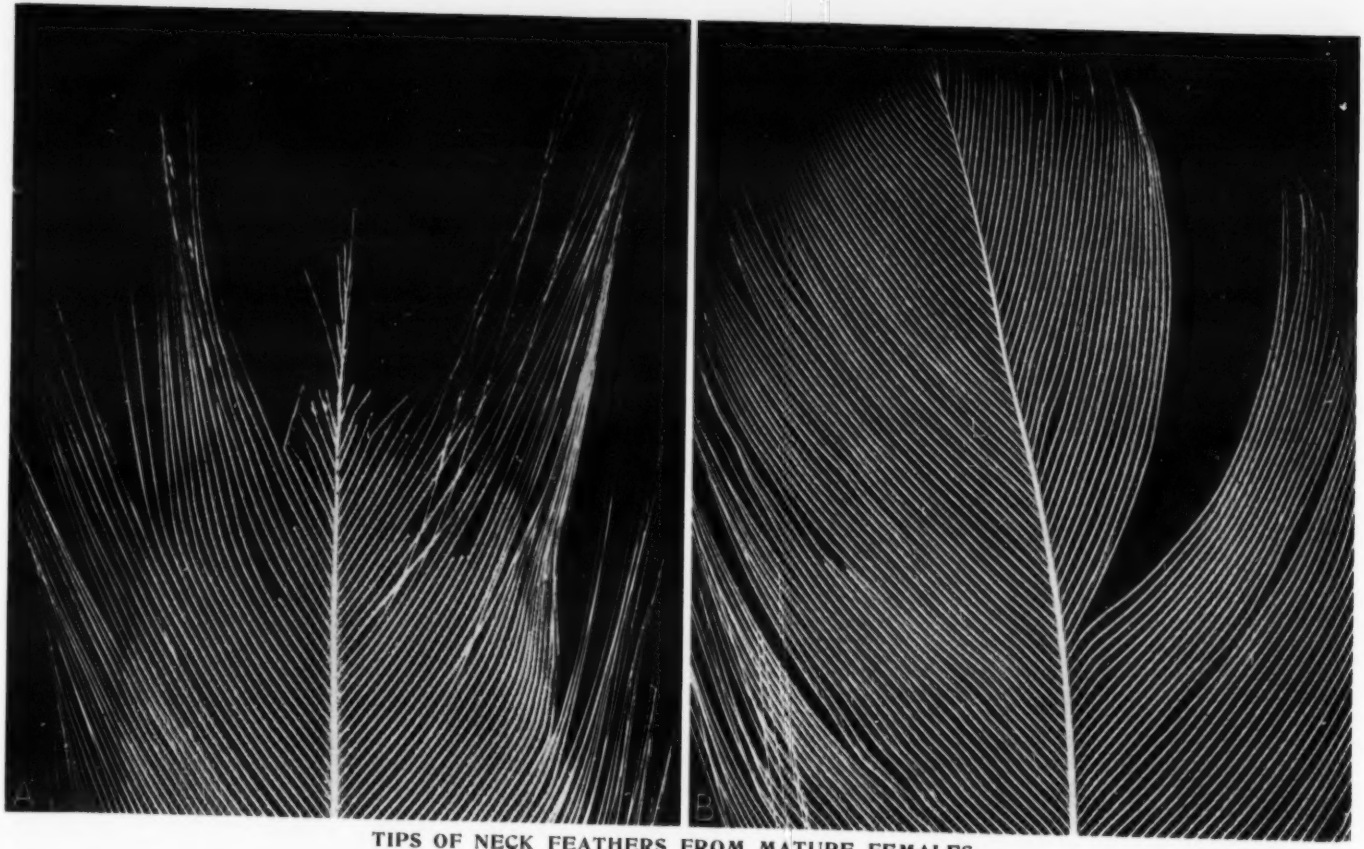
A. During Incubation

In the six matings of heterozygous males to normal females (Table I) the total number of naked chicks (246) among chicks hatched or dying after 18 days of incubation was slightly higher than that expected from the theoretical zygotic ratio (239). This proves that up to 18 days of incubation there is no greater mortality among naked embryos than in normal ones. During the last three days of incubation there is a marked difference, as is shown by the evidence from eight matings tabulated below. In order to restrict these data to the 19-21 day period, all unhatched eggs were broken for examination, and both normal and naked embryos which had died before 19 days were omitted from the class under consideration. The number of chicks hatched, plus those dead at 19-21 days, gives the total number alive on the eighteenth day. The following summary shows the mortality during the last three days in each mating, expressed in percentages, based separately upon the numbers of normal and naked chicks apparently alive on the eighteenth day:

	Died at 19-21 days	
	Normal %	Naked %
C 5919	17.6	65.0
C 5913	6.7	82.8
D 5976 (a)	6.9	46.7
D 5976 (b)	12.4	30.0
D 5983	4.3	33.9
D 5903	1.1	11.5
D 5977	10.0	40.0
D 5980	23.1	56.2
Weighted average:	8.5	46.7

The numbers in each mating are omitted but can be calculated, if desired, from Table I. The weighted averages are based on 755 normal and 287 naked chicks. In a brief earlier report of this condition by the senior author[6], it was stated that about three-quarters of the affected chicks die during the last three days of incubation. This was the case in the first two matings, but in later ones mortality among the naked chicks has been somewhat less, with an average of 46.7 per cent for all matings to date. While there is some evidence, as in the mating with ♂ D 5903, that when mortality in normal embryos is low deaths among the naked ones are correspondingly few, such a relationship is not always consistent. The highest mortality among naked embryos, 82.8 per cent, occurred in a mating with only 6.7 per cent mortality among normal ones. It seems probable, therefore, that while environmental agencies and genes affecting hatchability in general undoubtedly affect the hatchability of the naked chicks, there are also specific modifiers in some matings which particularly affect the naked chicks.

Those that die are full grown and in almost all cases have the yolk sac fully enclosed in the body. They do not appear to be different from those which hatch. It is probable, therefore, that most of the mortality occurs about the twentieth day of incubation.



TIPS OF NECK FEATHERS FROM MATURE FEMALES

Figure 4

The feather tips from a naked fowl (*A*) show the characteristic broken apical barbs and the elongation of barbules on a greater portion of their lengths than in normal feathers (*B*). The defects of the naked feathers are not due to a dietary deficiency, because feeding special diets had no effect, and transplants of normal skin to naked hosts grew a normal crop of feathers.

B. After Hatching

Mortality among naked chicks is higher during the first six weeks than in their normal siblings. Since many of the normal chicks were not raised in some of the matings, figures on mortality for all of those reported in Table I are not available. However, among sibling chicks which were raised under comparable environmental conditions mortality up to six weeks of age was as follows:

In 571 normal chicks: 13.1 per cent
In 150 naked chicks: 55.3 per cent

Mortality for both classes is somewhat high because these figures include chicks alive in pipped eggs which were helped out. Among them the death rate was unusually high. It is evident, however, that about half of the naked chicks which succeed in hatching succumb within six weeks. Since about 50 per cent die before hatching, this means that only about a quarter of the original zygotes survive to the age of six weeks after hatching.

Localization of the Defect

The question arises whether the abnormality induced by the *n* gene is localized in the feather follicles or is some general defect of metabolism. Martin and Gardner[7] reported that cysteine induced growth of hair in rats genetically hypotrichotic. This would indicate some metabolic defect, which these authors concluded was the lack of an enzyme necessary for breaking down "glutathione or other peptide linkages involving sulfur-containing groups" into amino acids. However, the fact that Roberts[8] was unable to confirm their results with cysteine and did show that skin from a normal rat would grow and maintain normal hair when grafted on a hypotrichotic one, indicates that the defect is in the hair follicles rather than in metabolism. A similar conclusion was reached by Crew[3] concerning the hairless mouse.

To measure the possible effects of cysteine on growth of feathers in the naked chicks, 7 of them, 5 days of age, were individually fed cysteine hydrochloride in aqueous solution as a supplement to an adequate stock ration. The amount given, 1 mg. per 3 grams of body weight, was greatly in excess of the dosages used by Martin and Gardner and by Roberts. It was kept reasonably constant by weighing the birds frequently and adjusting the amount of cysteine accordingly. After 21 days of this treatment the chicks fed cysteine did not differ from untreated controls in growth of feathers.

In a similar experiment using cystine with the same dosage as that for cysteine, no differences between eight experimental birds and their controls could be observed up to 26 days when the test was discontinued.

Mr. C. Dixon of this laboratory, who has successfully transplanted skin from naked donors to normal hosts and *vice versa*, finds that in persistent grafts the growth of feathers is the same as in the donor, regardless of the host. This shows that the mutation affects primarily the feather follicles.

Other Effects

Because of the high mortality before and after hatching it has thus far been difficult to obtain enough naked chicks of one age to permit reliable comparisons of them with their normal siblings in respect to growth, egg production, and other measures of physiological efficiency. Now that naked males have been secured that difficulty can be overcome. At hatching 18 naked chicks had an average weight of 39.8 grams compared with 40.55 grams for 18 normal siblings. At six weeks naked chicks and normal siblings had the following average weights in grams:

		♂♂	♀♀
Normal:	10 ♂♂, 8 ♀♀	466	427
Naked:	3 ♂♂, 7 ♀♀	515	392

Since the weaker naked chicks died and are not included, these figures merely indicate that growth in the best of that group is as good as that of normal chicks. Ten naked females for which records

are complete began to lay at a mean age of 178 days, with first eggs weighing on the average 45 grams. While these figures indicate that the naked birds are not radically different from other fowls, more extensive data on these points are desirable.

Discussion

Although different degrees of genetic hairlessness have been reported in the rat, house mice, deer mice, rabbits, cattle and swine, so far as the writers are aware this is the first case of generalized lack of plumage proven hereditary in birds. Professor L. J. Cole of the University of Wisconsin has informed the writers that he has some entirely featherless pigeons which are now being submitted to genetical analysis.

Aldrovandus[1] illustrated a nearly naked fowl which he labelled without further description as *"Gallina ferè petrificata,"* but his specimen differs from those reported in this paper in having the quills of the wing and tail feathers well developed, as are the shafts of feathers in other regions, but without normal barbs. It resembles more the "porcupine" mutation in pigeons reported by Cole and Hawkins[2] and is reproduced in the paper by those authors.

The naked condition differs from that in bare-neck or naked-neck fowls in having normal pterylosis but abnormal development in the feather follicles. Greenwood[4] has shown that the bare-neck condition, which is caused by an autosomal dominant gene, arises from restriction of the feather tracts in the anterior region of the body, and that where follicles are present normal feathers develop. The bare-necks lack down feathers and semiplumes in the apteria in all parts of the body, but the naked fowls may have almost normal feathers in the apteria particularly in the ventral and abdominal regions.

Embryos lacking down, or most of it, are occasionally found during the examination of eggs which have failed to hatch. The occurrence of three such embryos in related stock was reported by Serebrovsky and Petroff[9], who ascribed these abnormalities to genetic factors. However, Warren's[10] report of nongenetic complete downlessness shows that such an abnormality may arise entirely from extrinsic causes. Reduction of the down is not uncommon in embryos from fowls on diets deficient in riboflavin and in chondrodystrophic embryos, being most marked in both cases at about 13 or 14 days of incubation.

Naked chicks of about five weeks resemble somewhat the five-months-old birds illustrated by Warren[11], which were apparently homozygous for the autosomal dominant character, flightless. While the extent of the feathering is about the same in both, the flightless stock have defective beaks and toe nails, and the naked chicks do not. The former apparently have normal down at hatching. The flightless birds did not attain sexual maturity as the naked ones do. Moreover, at five months most of the naked chicks are much better covered than the two flightless specimens illustrated by Warren.

During the past year there appeared in the press a picture of an entirely nude chicken photographed by Mr. William LaVarre at Pisac, about twenty-five miles from Cuzco, in the mountains of Peru. In a personal communication to the senior author Mr. LaVarre has kindly provided additional information. Over fifty similar chickens were seen by him in the same district and he was sure that none had been plucked. The altitude is 12,000 feet, and the temperature between 20 and 60° F. The natives did not consider the naked birds unusual and stated that many years ago the wind had blown the feathers off many of their chickens, which subsequently produced featherless progeny. While this idea is open to question, it seems probable that there is a hereditary condition in Pisac which may be the same as that reported in this paper. The bird in Mr. LaVarre's photograph is considerably more naked than the adult stock thus far raised here. Its feather follicles seem normal in extent and distribution.

The genetically naked chickens discussed in this paper should not be con-

fused with those individuals extremely slow in feathering which are not uncommonly found in flocks of Rhode Island Reds, Plymouth Rocks and other heavy breeds, particularly in strains slow to mature. These have normal down at hatching and when they finally feather out are indistinguishable from normal chickens.

This new character should prove useful for clearing up some of the present uncertainties about the map of the sex chromosome in the fowl. For this the senior author[5] has recently suggested an arrangement different from those previously proposed. However, since of the seven genes which have thus far been assigned to that chromosome, five have effects measurable only in the plumage, the shortcomings of the new character, even apart from its lethal action, are obvious.

Freaks and mutations in poultry are reported continuously in the daily press. Many of the latter have been adopted as breed characteristics and others have been studied by geneticists. Since hereditary absence of feathers has not previously been established during the past 35 years of active genetic investigations with the fowl, and especially since sex-linked characters are particularly conspicuous, it seems probable that the mutation reported in this paper is of recent origin.

Summary

A hereditary character, naked, reducing the down of chicks and the plumage of adults is described. Pterylosis is normal, but the feathers do not erupt properly from their follicles. Remiges and rectrices are few or lacking. Naked fowls are easily distinguishable from normal ones at all ages.

This condition is caused by a sex-linked recessive gene, *n*, which on the average is lethal to about half of the affected chicks during the last two or three days of incubation. Among naked chicks hatched, mortality was 55 per cent during the first six weeks of life. Homozygous males are no more affected than hemizygous females.

Cysteine and cystine had no effect on the growth of feathers when fed in comparatively large doses. Skin transplants indicate that the defect is localized in the feather follicles.

Other conditions reducing the extent of the plumage in the fowl are briefly discussed and it is suggested that the naked mutation is of recent origin.

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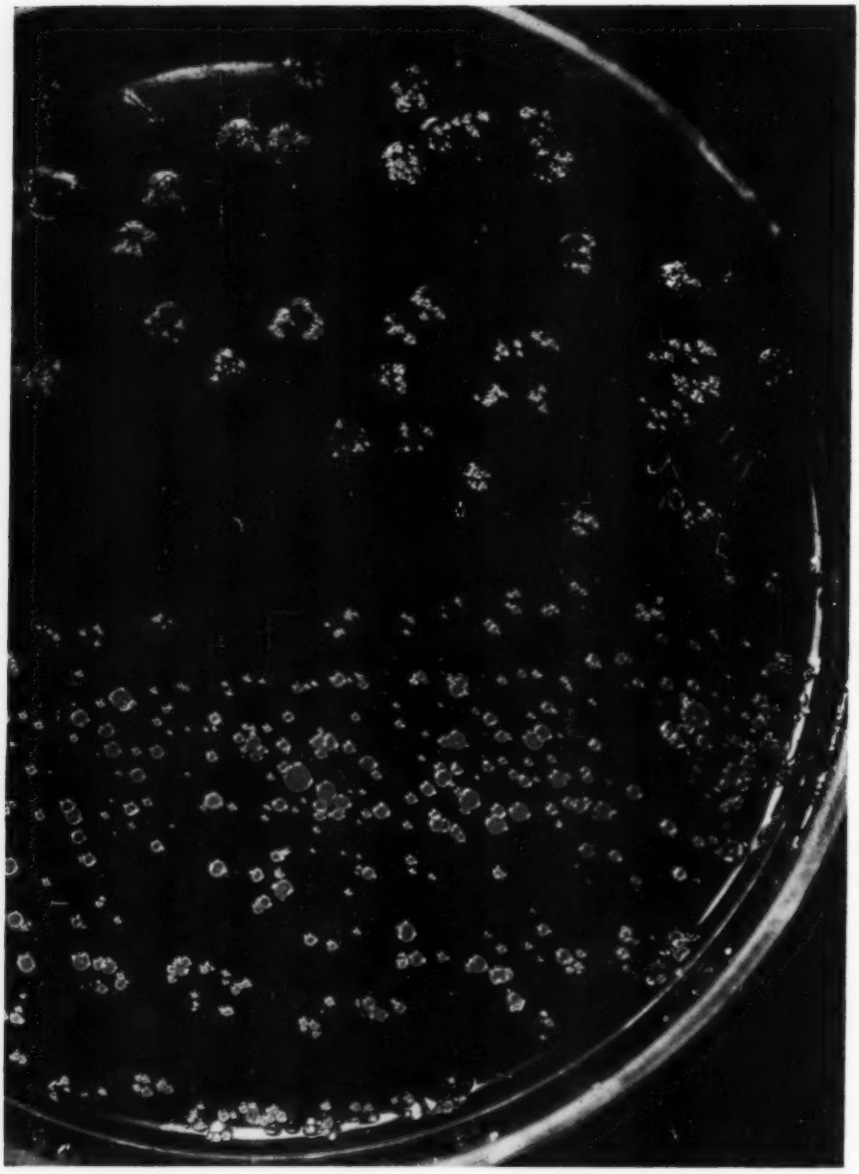


BACTERIUM COLI MUTABILE

Figure 5

Growth of Bacterium coli mutabile on a plate of lactose-indicator agar. All colonies are colorless the first 24-48 hours (dark in the photograph), and no irregularities can be seen. Shortly, where colonies are many, colored (light) points arise on and among the crowded colonies. On isolated colonies secondary colored colonies also soon appear, indicating lactose fermentation. Propagation from colored "points" or "secondaries" gives the promptly fermenting line, from colorless colonies or parts thereof gives delayed fermentation with the production, on plating, of the same cycle of "mutating" colonies.

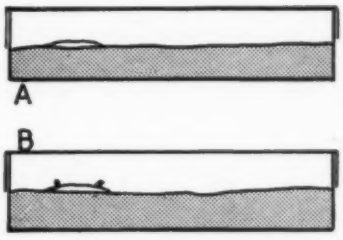
A NEW "MUTATION" IN THE COLIFORM GROUP OF BACTERIA

LELAND W. PARR
George Washington University

MOST bacteriologists know the *Bacterium coli mutabile* first described more than thirty years ago by Neisser and by Massini. When this organism is seeded on a lactose-indicator plate colorless colonies develop indicating the non-fermentation of lactose. When such plates are held for a few days tiny colored secondary colonies appear on the colorless colonies already present. (Figure 6). The color of these secondary colonies indicates that they are fermenting lactose, which the "primary" colony was not able to do. If such a "mutated" colony is carefully touched with an inoculating needle, avoiding contact with the primary colony on which it rests, the inoculum so secured will, on transfer to lactose broth, produce prompt and typical lactose fermentation and when this broth is plated on lactose-indicator agar colored colonies result.

On the other hand, if the primary colony is touched with a needle before any secondary colonies have appeared on it, or if a part of the original colony, which does have secondaries, is so "fished" as not to touch the secondary growth, this inoculum, in lactrose broth, produces no immediate fermentation and if such a broth be plated, one again obtains colorless colonies, lactose negative, on which in time colored, lactose-fermenting papillae or secondary colonies will appear.

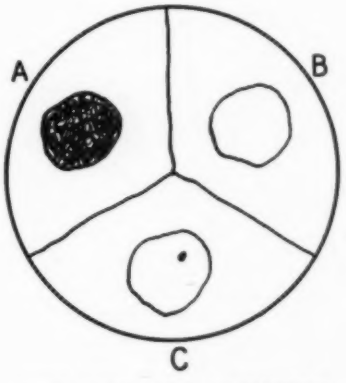
It is of interest that no one has ever obtained from such a "mutating" colon bacillus a growth which fails utterly to ferment lactose. Only the lactose-fermenting pure line and the "continuously sporting" lactose-defective, but not negative, strain seem to exist. Certain workers have even started with



COLONIES IN SECTION

Figure 6

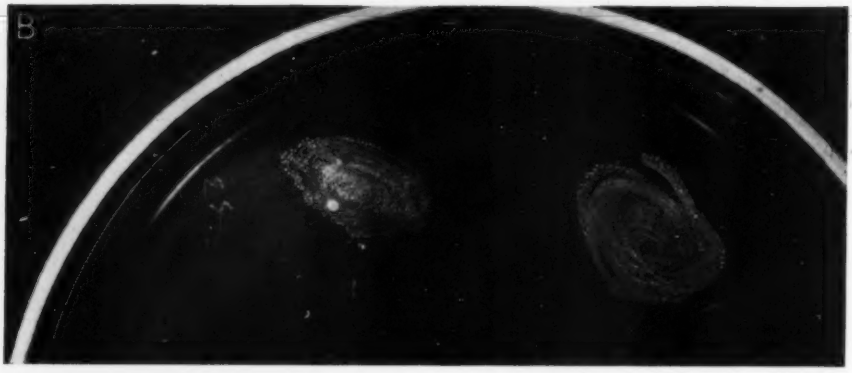
Section through Petrie dishes of colonies of *Bacterium coli mutabile* on lactose-indicator agar in (*A*) 24 hours and (*B*) after five days. A "bird's-eye view" of a plate containing many such colonies is shown on the opposite page (Figure 5).

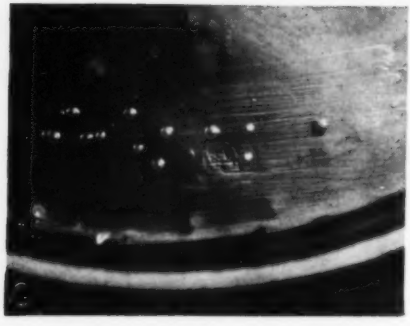


THE "CITRATE" MUTATION

Figure 7

"Bird's-eye" view of Simmons' citrated agar plate showing growth (*A*) of the typical citrate utilizers, the medium changing color; (*B*) of *Bacterium Coli* with no citrate utilization; and (*C*) of the citrate "mutation" which suddenly "acquires" the ability to utilize citrate.





A NEW "MUTATION" IN BACTERIA

Figure 8

A—(at right) A citrate positive colony produced by streaking a few drops of the culture on a sterile plate. The white border indicates active growth; (center and left) smears on citrate showing "mutating" colonies. The "mutations" tend to be more frequent around the edge of the streaking,—and they are not due to contamination. B—(right) A citrate negative colony; (left) a smear on citrate with one "mutant" colony developing. *C*—A large smear on citrate on which twelve "mutant" colonies have developed. This "mutation" involves a profound change in the metabolic economy of the organism, which enables it to "digest" a chemical compound which the parent form is unable to utilize, and which would probably not be encountered by the organism in its natural environment.

single-celled cultures and have obtained the same results.

Since these phenomena were first reported by Neisser and Massini, a number of workers have reviewed the evidence, and various hypotheses have been advanced in explanation. "Mutation" and "Mendelian segregation" of "duplicate factors" have been alleged,—though the absence of chromosomes in bacteria leaves us with no known mechanism which would control the orderly distribution of genes in the true Mendelian sense. Since the behavior of *mutabile* has been practically unique, it has rightly been considered something of a "special case." It is therefore a pleasure to add what seems to be another comparable instance of the same phenomenon, which has certain additional novel features.

In recent years the utilization of sodium citrate as a sole source of carbon by coliform bacteria has been one of the most widely used and most favorably looked upon of the tests used in the study of this group. *Bact. coli* does not utilize citrate, whereas *Bact. aerogenes, Bact. cloacae,* and *Bact. Freundii* (coliform intermediate) do. Simmons' citrated agar has been used in all of our work for this test. When an inoculum from a culture of *Bacterium coli* is spread on the surface of a Simmons' citrated agar plate no growth occurs, either at once or within a period of weeks. On the other hand the citrate utilizers *aerogenes, cloacae,* and *Freundii,* grow promptly producing less growth than on plain agar, it is true, but distinct growth nevertheless and the growth is accomplished by a change in color of the medium from the original green to blue.

Some five years ago we first encountered a coliform culture which when spread on citrate produced at first absolutely no growth but after three or four days a distinct colony appeared on the area later followed by one or two others. It must be remembered that the original inoculum contained thousands of bacteria. The number of bacteria in that inoculum capable of developing on citrate was therefore relatively a very small number. These "mutants" were, of course, checked to prove they were not contaminations and subsequent work has amply demonstrated that the phenomenon mentioned does occur. In a check of our records for over 5,000 strains of coliform bacteria isolated from fresh and from stored fecal specimens we find we have encountered this "citrate spotting" but 29 times. The "mutation" is, therefore not very common. (Figure 7.)

This citrate "mutation" has recently been given considerable study by methods and with results which will be given in detail elsewhere. Suffice it to say that by utilizing such "sporting" strains it has been possible to derive in pure and apparently stable culture (*a*) a prompt and typical citrate utilizing strain, (*b*) a strain entirely negative to citrate, and (*c*) the "mutating" parent strain which appears to carry on as such indefinitely and from which, almost at will, can be derived the two pure lines (*a*) and (*b*) mentioned above.

These three lines have been found to breed true under all conditions and over a long period of time. Serial transfer through milk, lithium chloride broth, and colchicine alkaloid broth* mediums did not result in alteration of the strains with respect to citrate. Neither did strains long kept in broth mediums without transfer show any change in citrate reaction when tested following such ageing. The three lines are, apart from citrate utilization, biochemically identical and all three react to approximately the same titre with antisera prepared from the original strain. The citrate variation is independent of the lactose variation of the *Bacterium coli mutabile.*

In addition to their general biological

*This broth was used to test the possibility that the profound effect which the alkaloid has on mitosis in higher forms might result in increased variability in bacteria. The results were negative.

interest such phenomena emphasize the need in bacteriological taxonomy for "lumping" rather than "splitting" species. The citrate "mutation" gives three lines,—(a), (b), and (c) to work with, whereas the lactose 'mutation" has never yielded but two. Such findings, further, give some hint as to interrelationships within the coliform group. Finally, the method offers a different approach to the mutation problem than has heretofore been generally used, for in dealing with lactose "sports" the lactose has usually been carried in mediums on which the strains grow whether lactose is utilized or not.

◆

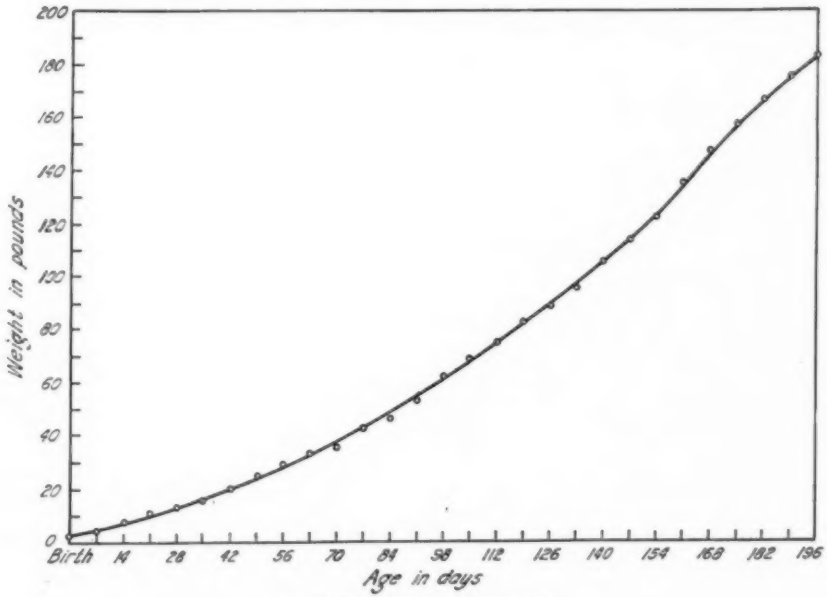
Talking Social Sense in the Language of Size

TWO views are commonly held about mathematics. One comes from Plato. This is that mathematical statements represent eternal truths. Plato's doctrine was used by the German philosopher, Kant, as a stick with which to beat the materialists of his time, when revolutionary writings like those of Diderot were challenging priestcraft. Kant thought that the principles of geometry were eternal, and that they were totally independent of our sense organs. It happened that Kant wrote just before biologists discovered that we have a sense organ, part of what is called the internal ear, sensitive to the pull of gravitation. Since that discovery, the significance of which was first fully recognized by the German physicist, Ernst Mach, the geometry which Kant knew has been brought down to earth by Einstein. It no longer dwells in the sky where Plato put it. We know that geometrical statements when applied to the real world are only approximate truths. The theory of Relativity has been very unsettling to mathematicians, and it has now become a fashion to say that mathematics is only a game. Of course, this does not tell us anything about mathematics. It only tells us something about the cultural limitations of some mathematicians. When a man says that mathematics is a game, he is making a private statement. He is telling us something about himself, his own attitude to mathematics. He is not telling us anything about the public meaning of a mathematical statement.

If mathematics is a game, there is no reason why people should play if they do not want to. With football, it belongs to those amusements without which life would be endurable. The view which we shall explore is that mathematics is the language of size, and that it is an essential part of the equipment of an intelligent citizen to understand this language. If the rules of mathematics are rules of grammar, there is no stupidity involved when we fail to see that a mathematical truth is obvious. The rules of ordinary grammar are not obvious. They have to be learned. They are not eternal truths. They are conveniences without whose aid truths about the sorts of things in the world cannot be communicated from one person to another. In Corbett's memorable words, Mr. Prynne would not have been able to impeach Archbishop Laud if his command of grammar had been insufficient to make himself understood. So it is with mathematics, the grammar of size. The rules of mathematics are rules to be learned. If they are formidable, they are formidable because they are unfamiliar when you first meet them—like gerunds or nominative absolutes. They are also formidable because in all languages there are so many rules and words to memorize before we can read newspapers or pick up radio news from foreign stations. Everybody knows that being able to chatter in several foreign languages is not a sign of great social intelligence. Neither is being able to chatter in the language of size. Real social intelligence lies in the use of a language, in applying the right words in the right context. It is important to know the language of size, because entrusting the laws of human society, social statistics, population, man's hereditary make-up, the balance of trade, to the isolated mathematician without checking his conclusions is like letting a committee of philologists manufacture the truths of human, animal, or plant anatomy from the resources of their own imagination.—HOGBEN, LANCELOT, Mathematics for the Million (pp. 27-28).

A NORMAL GROWTH CURVE FOR SWINE

N. R. ITTNER and E. H. HUGHES
University of California, Davis



HOW 457 PIGS GREW UP

Figure 9

Average growth rate of 457 pigs, from birth to 196 days of age, in the University of California herd. The maximum rate of gain was at an age of about six months, after which the weekly gains began to decrease.

TO evaluate the results of critical genetic and nutrition studies a normal growth curve for swine is essential. While such curves have been reported by Russell[4] and McKenzie,[3] an examination of Table I, where similar data are presented, demonstrates that additional information is needed. The data are similar from birth to 10 weeks of age, but vary considerably from that point to an age of 28 weeks. The differences are probably due to genetic, environmental, and nutritional factors. Data collected at this station are graphically represented in Figure 9. The material presented is taken from more than 5,000 weighings and represents over 450 individuals, 171 before and 286 after weaning. The figures for each week shown represent a minimum of at least 150 weighings. The data for pigs before weaning are based on normal individuals from sows on normal diets. After the pigs were old enough to eat, they were allowed free access to feed in creeps except for short periods following vaccination, which was usually done when the pigs were five or six weeks old. The data for the animals after weaning were taken from pigs fed such diets as these:—barley 88 pounds, tankage 7 pounds. linseed meal 3

pounds with pasture at will; barley 84 pounds, skim-milk powder 3 pounds, tankage 3 pounds, fish-meal 3 pounds, alfalfa-meal 5 pounds, some had free access to pasture, some not; barley 1 part, skim-milk or buttermilk 3 parts fed 2 and 3 times a day, some with, some without, pasture; barley 3 parts, raisins 1 part, skim-milk 12 parts with and without pasture; barley 1 part, whey 3 parts without pasture; barley 9.2 pounds, alfalfa-meal 8 pounds with and without pasture. In most of the diets oyster-shell flour and salt were included. The pasture was either alfalfa or Sudan grass.

An enormous amount of data were available but to present it logically was not easy. To obtain the birth weight was not difficult because all the pigs were weighed when born or within 10 hours thereafter, and the average was taken as the mean birth weight. To obtain a curve of increasing weights, it was decided to group the data and plot the curves by weeks. To do this, the seventh day from birth was taken as the starting point, and the weights of all the animals for 3 days before and 3 days after were added to those of the seventh day. This figure was divided by the number involved which gave the average weight for the seventh day. This was continued and from it the data in Table I were developed.

When plotted (Figure 9), the data show a smooth curve, which was expected from the number of weights and individuals represented. The curve shows a gradual acceleration to a point where the pigs weighed about 160 pounds. From that time the curve begins to slope the other way. The curve is not unlike that shown by Russell[4] for the pig, except that it is slightly steeper, nor that of Donaldson[1] and Hogan[2] for the rat.

That the growth curves developed by Russell[4] and by us should differ somewhat after the first 10 weeks (about weaning time) is not surprising. There were probably environmental differences, such as temperature; the diets fed no doubt varied in composition and quantities fed; the genetic constitution of the animals involved was similar, yet their feeding ability and growth factors were probably unequal.

Summary

A growth curve for young, growing pigs is presented. The data was taken from the records of the California Station herd.

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Table 1. Comparison of Two Sets of Data (bi-weekly)

Age Weeks	U. S. Dept. of Agri.* Pounds	Calif. Exp. Sta. Pounds
Birth	2.6	3.2
2	8.5	7.5
4	13.7	13.1
6	19.9	20.0
8	27.7	29.4
10	36.2	35.8
12	42.9	46.8
14	51.1	61.4
16	58.1	75.4
18	70.9	89.4
20	83.1	105.6
22	97.6	121.8
24	106.5	147.5
26	121.8	166.8
28	133.0	182.5

*Taken from Missouri Agricultural Experiment Station Research Bulletin 96.

GENETICS OF DYSDERCUS

I. Behaviour of a Melanic Form of *Dysdercus Mendesi* Bloete

LUIZ O. T. MENDES
Seção de Entomologia, Instituto Agronomico
Campinas, S. Paulo Brasil

THE genus *Dysdercus* belongs to the order Heteroptera, family Pyrrhocoridae. Its genetics is almost unknown, only one pair of factors (*R-r*) having been investigated.[2]

Since January, 1936, I have been carrying out investigations on several species of this genus, known as cotton pests in the State of Sao Paulo. In the course of these studies I frequently noticed conspicuous variations in the general body color and patterns. Considering the ease of handling, the relatively large offspring of one female and the short life cycle (about 40 days), *Dysdercus* proved to be excellent material for genetical studies; the relatively large size of the adults and nymphs makes it convenient class material for demonstration.

Among the variations that attracted my attention, was a melanic form in the species *D. mendesi* Bloete. So far as I know, melanism has not yet been studied genetically in any group of the order Heteroptera. Among other orders, mostly Lepidoptera, it has been investigated, sometimes being a recessive and sometimes being a dominant character.

Occurrence of the Melanic Form

In April, 1936, my attention was called to several nymphs that were somewhat different from the others belonging to the same family and batch. These developed into adults of general black color, while from their normal sisters reddish adults emerged.

Turning back to my notes on the genealogy of these insects I found that they originated from a cross of two wild (reddish, normal) insects: the male used in this cross had also been mated to another wild female and here again the melanic form appeared in the offspring.

I suspected that the wild male must have been heterozygous for the factors determining the melanic form. It was not a case of mutation in the laboratory, as the above mentioned male was caught outside in the field. Melanic individuals were also found in nature, although very rarely. Once a melanic male has been raised from a batch of nymphs collected in the field; besides, from May to November, 1936, (6 months) I found in the field three melanic adults; two males and one female.

Before the isolation of this family, I tried to cross melanic individuals *inter se*, but no results were obtained.*

Differences Between the Normal, Melanic and Intermediate Forms

There are no visible differences between nymphs of the melanic and wild forms during the first instar; the first symptoms of differentiation appear in the second; in the third the differences become more visible and in the fourth and fifth the melanic individuals can be separated quite easily from the others. Colors of the different bodyparts of nymphs, during the fifth instar are predominantly red or yellow; comparable nymphs of the melanic form are predominantly black. Detailed comparisons of color of the nymphs

* In 17 out of 21 attempts to mate the melanic individuals no copulation at all occurred; in three out of the four cases of copulation no eggs were obtained; in only one instance eggs were obtained after copulation, but all were sterile. The occurrence of sterile eggs from virgin females of *Dysdercus mendesi* Bloete, *D. ruficollis* (L) and *D. honestus* Bloete has been observed frequently.

and adults are presented in the supplementary tables.*

During the fifth instar the adult pigmentation of the melanic forms is clearly evident. The normal nymphs of the fifth instar, like normal adults, are predominantly reddish in color, with yellowish markings. The melanic nymphs are predominantly black, as are the adults from the mutant forms.

The adults of the melanic form are easily distinguished from the normal ones by the general body color; the normal has a reddish color; the other is black.

Genetic Analysis of Melanic Mutant

As soon as adults appeared in the cages, males and females were separated from the nymphs and from each other, in different rearing cages, in order to obtain virgin females for our studies.

Crossings. Six different crosses were made, according to the technic described: 1. *Normal* × *Normal* — 34 fertilized females gave 4,120 eggs; all the nymphs examined (2,040) were Normal. Table III* itemises these crosses.

2. *Normal* × *Melanic* — Fifteen crosses were made in either way (*Normal* × *Melanic* and *Melanic* × *Normal*). The resulting 1,277 nymphs examined were, as expected, all intermediate. (Table IV.*)

3. *Intermediate* × *Intermediate* — In 22 crosses made between heterozygous individuals, 1,457 nymphs were classified. These gave a ratio of 1,107 Normal *and* Intermediate to 350 Melanic (The details are given in Table V*). In many of the progenies the ratios were very satisfactory (approximately 3 apparently Normal to 1 Melanic) but in a few progenies a certain deviation was observed. However, this discrepancy proved not to be statistically significant according to Fisher's method for "goodness of fit," the actual ratio of 1,107 :350 being a good 3 :1 ratio ($\chi^2 = .742$).

In seven out of the 22 families the offspring were classified into the expected three phenotypes, the observed ratio 163 normal : 328 intermediate : 151 melanic (Table VI*) is not statistically different from the expected 160.5 :321.0 : 160.5 ($\chi^2 = .753$).

4. *Normal* × *Intermediate.* — Nine crosses were made in both directions (*Normal* ♂ × *Intermediate* ♀, *Intermediate* ♂ × *Normal* ♀). Of 695 nymphs classified, 336 were normal and 359 were intermediate, where 347.5 of each class were expected. There is no significant difference between the observed and the expected ratios ($\chi^2 = .760$).

5. *Intermediate* × *Melanic* — An equal number of melanics and intermediates was expected in this cross. The observed ratio, based on the results of 13 crosses in both directions, is not very close to it; we had 641 intermediates : 589 melanics (instead of 615 :615). The chi-square, however, shows that it is a reasonbly good 1 :1 ratio ($\chi^2 = 2.196$).

6. *Melanic* × *Melanic* — All the 1,765 progeny obtained in 15 crosses were entirely constituted of melanic insects, as was expected (Table IX*), if melanism is controlled by a recessive gene.

Discussion and Conclusions

By breeding tests it has been proved that the melanic form of *Dysdercus mendesi* Bloete, found in this laboratory, is determined by a single pair of recessive factors. The normal form is incompletely dominant over the melanic. For the pair of factors studied the author proposes the symbols *mm.*

The melanic form (*mm*) here mentioned is found relatively seldom in the cotton fields near Campinas (S. Paulo)

* This article was accompanied by nine detailed tables setting forth the color-distribution of normal and melanic forms, and tabulations of observed and expected results of all the crosses tested. These tables have been afforded supplementary publication as Document No. 1144 of the Documentation Institute. Copies of these supplementary tables can be obtained on microfilm (25 cents) or as photoprints (75 cents) by ordering from the American Documentation Institute, 2101 Constitution Avenue, Washingon, D. C. Order Document No. 1144, remitting amount to cover service desired.

where the normal (typical) form is very abundant.

A careful analysis of the results presented in Tables III to IX shows that:

a. There is an excess of heterozygous individuals over homozygous in all the crosses where heterozygous (*Mm*) and homozygous (*MM* or *mm*) individuals are segregating.

b. A lower fertility of eggs seems to exist in the following crosses: *MM* × *MM* and *mm* × *mm*; the highest average of fertility has been obtained in the crosses *MM* × *mm* and also in *Mm* × *mm* and *MM* × *Mm*.

2. The actual and expected results of all the crosses made are presented in tables which have been afforded supplementary publication as Document No. 1144 of the American Documentation Institute.

Summary

1. The author, who is investigating *Dysdercus mendesi* Bloete as a cotton pest, since 1936, found a melanic form of this species heretofore unknown and of very rare occurrence in the field. The author concludes after a detailed genetical analysis that the character is determined by a single pair of genetical factors (*mm*) recessive to the normal type (*MM*). The heterozygous individuals (*Mm*) are phenotypically intermediate between the *melanic* and the *normal*; it is therefore a case of incomplete dominance.

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✦

A SINGLE CHROMOSOME EXPLANATION OF ROBERTS AND QUISENBERRY'S BRAHMA × BARRED ROCK MOSAIC

S. S. Munro

Central Experimental Farm, Ottawa, Canada

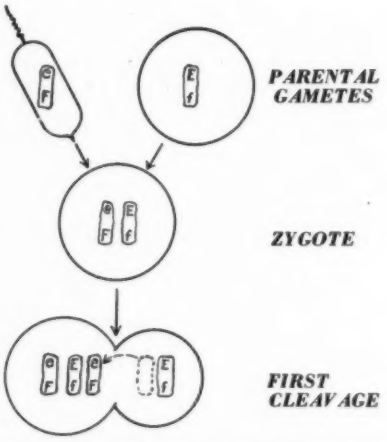
RECENTLY, while working in the laboratory of Professor F. A. E. Crew, the writer became interested in mosaicism and gynandromorphism in birds and thanks to the generosity of that eminent biologist had the rare privilege of intimately collaborating with him in a study of the many specimens which he had accumulated at the Edinburgh Institute. Due principally to the fortunate occurrence of one very unusual specimen (case *D* in the published report[1]) we were able not only to show that gynandromorphism in the domestic fowl could occur independent of aberrations of the sex chromosomes but also to suggest that the outward expression of certain autosomally located genes (at least the flesh color alleles *W*, *w*) depends on gene balance. Although the presence of a single dominant white flesh allele *W* masks the presence of a single yellow allele *w*, so that a heterozygous individual appears white fleshed, non-disjunction or other autosomal aberration which results in the presence within the same cell of two recessives (*ww*) and one dominant (*W*), will result in yellow flesh. In other words $W > w$ but $W < ww$. Only by such assumption could we explain our critical case *D* and remain in hypothetical harmony with other simultaneously reported white-yellow flesh mosaics.

While our explanation cannot as yet be accepted as final it is interesting to note that a similar resort to the gene balance theory, already firmly established in Drosophila, will satisfactorily explain the Brahma-Barred Rock mosaic reported by Roberts and Quisenberry[2] on a single chromosome basis providing the genes for feathered legs,

F, and extension of melanin pigment, *E*, are linked. A sperm from the Light Brahma father fertilizing an egg from the Barred Rock mother will produce an individual heterozygous for these two factors, *FfEe*. If these genes are located on the same chromosome, *F* and *e* would enter the zygote on the Light Brahma chromosome whereas *f* and *E* would enter on the Barred Rock chromosome. If, at the first division, the *Fe* chromatids failed to separate or after separation they migrated to the same pole of the spindle, one daughter cell contains two Brahma derived chromosomes each carrying genes *F* and *e* as well as one Barred Rock chromosome carrying *f* and *E*. Under these circumstances the left side of Roberts and Quisenberry's mosaic would be *FFfeeE* and the right side *fE*. According to our hypothesis such autosomal heteroploidy would produce a marked difference in skeletal size between the sides, probably between 10 and 15 per cent linear difference in long bone measurements; in addition the balance between *E* and *e* would be altered. The mechanism of genic action may be visualized in many ways, however, in this particular example it is convenient to consider that emanations originating from the *e* gene determine the threshold which the emanations from *E* must exceed before melanin can be deposited in the feather. When two *e*'s are present within the same cell, it is quite possible the increased threshold is beyond the attainment of a single *E*, in which case the feathers would lack pigment except in the hackle, wing and tail and would thus resemble the Light Brahma. Under these circumstances a bird which is *FFfeeE* on the left side and *fE* on the right would resemble in all respects the mosaic described by Roberts and Quisenberry.

The objection to this explanation is the fact that two assumptions must be made. First, that *F* and *E* belong to one linkage group and second that $E<ee$. So far as the author is aware no evidence either for or against either of these assumptions exists. However, a mosaic chicken is an extremely rare phenomenon. Thirteen cases, not including the Brahma-Barred Rock mosaic under discussion, are listed by Crew and Munro.[1] Each case involves only one pair of Mendelian characteristics other than size and each one can be explained satisfactorily on the basis of a single chromosomal aberration, either elimination or non-disjunction. However, in Roberts and Quisenberry's case, if the independence of *F* and *E* be admitted, one is forced to postulate either, as did the original authors, the loss of two independent autosomes one from each of the daughter cells, or, as Crew and Munro[1] suggested and what is more probable in view of the pronounced lateral size asymmetry, non-disjunction in the *F* autosome and elimination of the *E* autosome.

From the scientific standpoint, the simplest explanation is more likely to be the correct one. In view of the apparent rarity of chromosomal aberration



HOW THE MOSAIC MAY BE FORMED

Figure 10

Suggested single chromosome explanation of the Brahma-Barred Rock Mosaic involving linkage and gene balance. The heteroploidy induces lateral size asymmetry; $ee>E$ thus restricting melanin pigment on the left side making it phenotypically Brahma; *F* removed from right side leaves it clean shanked.

in the domestic fowl the writer submits that his new explanation, i. e., non-disjunction of a single autosome, notwithstanding the fact that it involves the corollary assumptions of linkage and gene balance is at least as likely as one involving aberrations in two independent chromosomes. The new explanation has the advantage of being open to proof or rejection. If subsequent research shows that *F* and *E* are linked, not only will it provide a single chromosome explanation of the Brahma-Barred Rock mosaic and establish a new linkage group, but it will also place the existence of genic balance in the fowl on an indisputable basis and thus justify the explanation of case *D* in our original paper.[1]

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ARTIFICIAL INSEMINATION IN RUSSIA

MILOVANOV'S recent book on artificial insemination* will be of great interest to animal breeders in all parts of the world. It is the fourth enlarged edition, and the changes and additions demonstrate how rapidly this technique is developing in Russia.

Artificial impregnation of farm animals, particularly sheep, and in lesser degree of cows, and horses has been practiced and developed in Russia more than in any other country. A special Artificial Insemination Laboratory has been established at the famous Live-Stock Farm and Station at Askannii Novy (on the shores of Black Sea), and the semen is prepared under auspices of this Laboratory called LIO in Russian, which Laboratory has dozens of branches, and under whose auspices men are trained to specialize in this work. Details regarding the extent to which the method is now used are given below. The Soviet Government is interested to make this method more universally used, even in isolated regions and by individual peasants who may not have large flocks or herds. In order to do so, a great many experiments were conducted to find means whereby sperm could be easily transported and remain viable for a considerable time. Recently Milovanov, one of the leading Russian workers in the field of artificial impregnation, developed a new method of preparing this material, so that it may be shipped in very easily handled, inexpensive capsules and also easily applied by anyone.

Milovanov calls his method "Geletanized Sperm," and he describes it in a 38-page pamphlet,† from which the following brief abstract is presented for the benefit of American specialists and stockmen.

The semen is extracted from the male animal daily or as needed by specially devised instruments. The semen is then diluted with a special formula which contains chemicals (these are given in the booklet) and with gelatin. After thorough dilution, this thickened material is placed in individual doses, and sent out in special capsules packed in cotton, and wrapping paper to the farms.

Instead of using expensive glass containers, capsules or others difficult to handle, Milovanov uses the stems of bulrushes or reed (*Arunda phragmites*).

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*Milovanov, V. K. ISSKUSSTVENOYE OSSEMENENIE SELSKO-KHOZIASVENNYKH JIVOTNYKH (Artificial Insemination of Farm Animals) Pp. 368; 227 illus. Selkhozgiz, Moscow, 1938.

†ISSKUSTVENNOYE OSSEMENENIE Selske—Khoziasvennykh Jivotnykh Jelatinirovannoi Spermoi—("Artificial Insemination of Farm Animals with Gelatinised Sperm"). V. K. Milovanov—Bulletin No. 23, of the Series on "New in Agriculture," published by Selkhozgiz—Moscow, 1938.

These are prepared, sterilized, etc., during winter, and are filled with the semi-hardened material, sealed, and properly packed during the breeding season.

Upon receipt of these "reed capsules," the farmer can very easily insert the capsules into the vagina of the animal by simply using a wooden or glass expeller which takes out the diluted gelatinized semen. Complete instructions with illustrations are given in the booklet. The results are reported to be remarkable, and this method is now recommended by the Soviet Commissariat of Agriculture. The booklet contains chemical and mathematical formulae, discussion of tests made, etc.

Extent of Use of Method

In Russia the practice of artificial insemination in animal breeding dates back to before the war. The Tzarist Government began using the method on its stud farms in 1909, and between 1909 and the beginning of the World War, 7,000 mares were artificially inseminated. No artificial insemination was done during the War and Civil War periods, but beginning with 1923, this work was revived, and during 1923-1925, 12,000 mares were inseminated. This number increased every year until in 1932, it grew to 182,000 mares. Due to the neglect of horses, and the growth of mechanical power (tractors), the number of inseminated mares decreased to 8,000 in 1936, but took a spurt again in 1937, when 53,000 were inseminated. This (1938) year, 120,000 mares were to be inseminated.

The artificial insemination of sheep did not start until 1928 (which by the way was the year that American Rams were first imported), and that year amounted only to 4,703. By 1931 the number of ewes reached 582,946, the following year it reached 1,615,400, by 1934 it was over two million, and 1936—eight milllions, last year—twelve millions, and this year, fifteen million ewes were to be artificially inseminated.

With cows, artificial impregnation was started in 1930, when 19,970 were impregnated. In 1931—185,000, next year 385,000. Then for a few years, the number dropped to 100,000-150,000 annually. In 1936, it rose to 500,000. In 1937 a million, and this year a million and a half cows were to be inseminated. The work with hogs and poultry is also extensive, but no figures are given.

It is also interesting to note a few figures, as to numbers of inseminations that Russian workers report to have done with one male animal. The following official records are given by Milovanov and other Soviet writers: Jacob Tzibulia, a member of Kolkhoz "Svobodni Trud" located on Arzirsk district of Ordjonokidze region, inseminated with the sperm of one Rambouilet ram—3,554 ewes. Mr. Petrov, in Orlov district of Rostov region inseminated 15,000 ewes with semen from Rambouilet ram No. 2103. In this case, record was kept on the 12 collective farms (kolkhozi) where the work was done, and out of 13,809 ewes inseminated artificially, there were born 14,944 lambs—twins more than making up for failures of the method. Another man, by name Petrov in the same region, inseminated 11,800 ewes with semen from one Rambouilet ram No. 880. Here also records were kept on 14 collective farms, and out of 11,256 ewes inseminated, there were born 918 lambs.

The average number of inseminations from one ram reached in 1936 in the Orlov district 5,600 ewes, and in a number of regions—1,088. The percentage of "takes" also increased in several regions from 83 per cent in 1932-33 to 96 per cent in 1935-1936.

The record with cows: one bull Molodetz of the Yaroslav breed inseminated 530 cows. In the Tagil breed, the bull Martika inseminated 1,450 cows.

About 25 per cent of ewes are now artificially inseminated in USSR, and about five per cent of cows, but equipment and techniques are being so rapidly improved and so quickly adopted that within the next few years this method will be still more generally used. At the present time, it far exceeds anything done in all other European and American countries combined. J. W. Pincus

THE MIDGET TOMATO

A New Gene Mutant

J. W. LESLEY
University of California Citrus Experiment Station, Riverside, California

MIDGET AND NORMAL TOMATO PLANTS

Figure 11

The plants are of the same age and received similar cultural treatment.

A NEW mutant type of tomato conspicuous for its small size and dense cushion-like growth habit appeared at Riverside in 1935, as a single plant from a seed of the variety Norton. This growth habit was due to the development of an unusually large number of axillary shoots. Adventitious

roots were especially common on stems above the ground, possibly on account of the dense shade resulting from the compact growth habit. The name "midget" was chosen because the stems, leaves, and flowers are much less than normal size (Figure 11). The green color of the stem and leaves is fully as dark as the normal.

The plant produced very few flowers in the field and these usually opened only after considerable elongation of the shoot had occurred distal to the inflorescence. Usually these flowers were quite abnormal and no pollen matured. No fruit was produced in the field. A plant from a cutting of the original midget flowered more freely in the greenhouse in winter, cymes of 2—4 flowers being common, but the anthers, which were especially small, contained extremely little pollen and no fruit set from self-pollination. After several failures, a single fruit set from cross-pollination with a race provided by Dr. J. W. MacArthur, University of Toronto, containing recessive genes belonging to six linkage groups. The fruit of the midget was scarlet as in the original variety Norton, much cracked, and only about one-fifth normal size. It contained nine seeds from which three F_1 plants were obtained. These showed none of the midget characteristics and were self-fertile.

The F_2 consisted of normal plants and midgets. Three F_2 sowings were made of seed from one F_1 plant and subsequently, two F_3 sowings from one heterozygous normal F_2 plant, and one F_3 from another F_2 plant which was probably not heterozygous for midget. The populations in which segregation occurred contained only 15 per cent instead of 25 per cent expected from monogenic segregation. Table I shows that the proportion of midgets in the F_2 and F_3 populations was very variable but always less than the expected proportion for a simple recessive character. Since this proportion tends to decrease as the percentage of germination decreases, midget is probably less viable than the normal. The observed numbers are consistent with the hypothesis that it is a simple recessive of low viability.

TABLE I. Progenies from selfing normal F_1 and F_2 plants originating from crossing normal ♀ X midget ♂

Parent	Generation of progeny	Normal	Midget	Per cent midget	Per cent seed germination
C3bb-1	F_2	247	52	18	91
C3bb-1	F_2	71	19	21	83
C3bb-1	F_2	36	5	12	76
C3bb-1-1	F_3	71	2	3	68
C3bb-1-1	F_3	67	1	1	48
C3bb-1-2	F_3	77	0	--	71

◆

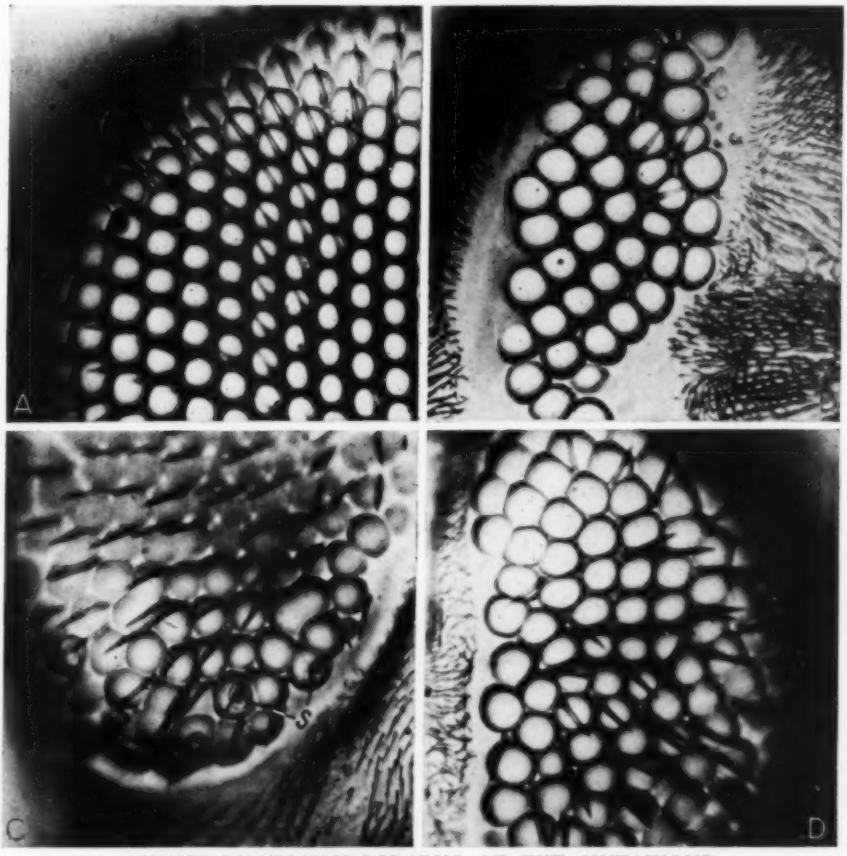
Anthropoid Blood-Relationships

The reactions obtained with a variety of anti-M and anti-N fluids, confirm the original observation of Landsteiner and Levine that the blood of all chimpanzees contains agglutinogens similar to but not identical with the human agglutinogens M and N. Moreover, it could be shown that M agglutinogens are also present in the blood of gibbons, orang-utans and certain species of lower monkeys. The degree of resemblance betwen the M agglutinogens in monkey or ape blood and human blood shows a certain degree of correlation with the position of the monkey or ape in the evolutionary scale.

From the behavior of different anti-M sera in tests on monkey blood, the existence of a number of qualitatively different M agglutinins may be assumed, yet all the sera behave alike in tests on human blood. Which M agglutinins will be formed when rabbits are immunized with human M blood evidently depends upon constitutional attributes of the rabbit.—Dr. A. S. Wiener in *Jour. Immun.* 34:11-18. 1938.

THE "SUMP" METHOD FOR DROSOPHILA MORPHOLOGY

HIDEO KIKKAWA
Zoological Institute, Kyôto Imperial University

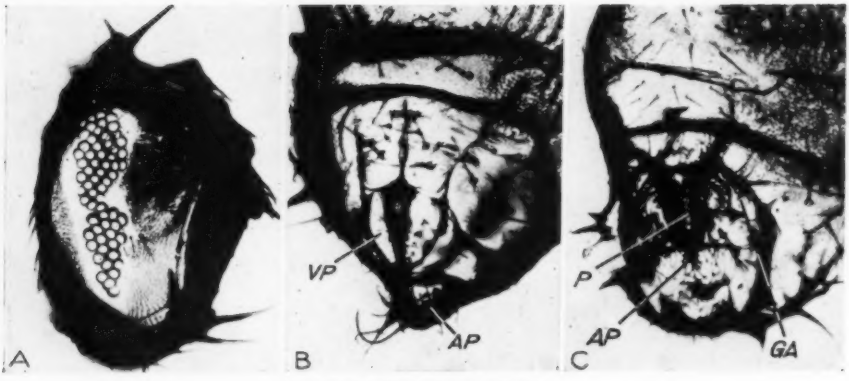


"SUMP" PHOTOMICROGRAPHS OF EYE MUTATIONS

Figure 12

Eye surface of (*A*) wild type *Drosophila melanogaster;* (*B*), Bar (I-57.0); (*C*), roughoid (111-00). Note the specks (*S*) on the ommatidia; *D*, eyeless (IV-0.0). Enlarged about 360×.

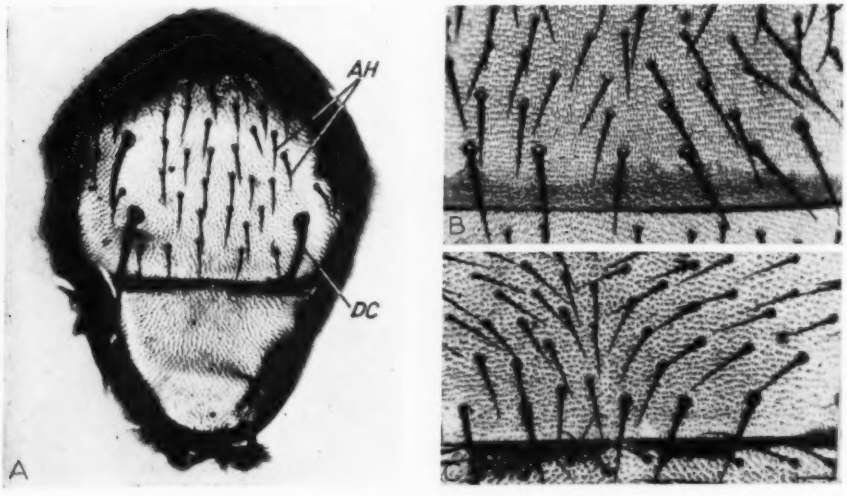
IT will be of great advantage for Drosophilists if there is some convenient method for examining minute surface structures of the flies. Two current methods are in general use: (1) clarifying the object with some agent such as concentrated caustic alkalies (saturated KOH or NaOH) or lactophenol solution (lactic acid 1 + glycerine 2 + phenol 1 + water); or (2)



ANATOMICAL DETAILS

Figure 13

A—Total photograph of Bar eye of *D. Melanogaster*. The character gets its name from the arrangement of the few functional ommatidia (eye facets) in a vertical band across the eye. *B-C*—external genital apparatus of *D. melanogaster*, female (*B*) and male (*C*). *AP*=anal plate, *VP*=vaginal plate, *GA*=genital arch, *P*=penis. 90×.



BRISTLE DETAILS BY "SUMP" METHOD

Figure 14

A—Thorax of *forked* individual of *D. melanogaster*. Dorsocentral and scutellars are the original bristles which were left in the celluloid film. *AH*=acrostichal hairs; *DC*=dorsocentral. *B*—Bristles and hairs on abdomen of normal *D. virilis; C*—same on *irregular* individual (IV-76.0) about 120×.

using a microscope with a special outfit, such as Leitz's Ultropak.

These methods, however, are unfit for taking photographs or for preparing lantern-slides. The "Sump" method which I am going to describe in this communication is simple yet very convenient especially for such purposes, and worthy of special recommendation. The method was invented in 1928 by Dr. J. Suzuki, SUMP being the combination of the initial letters of Suzuki's Universal Micro-Printing. This technique has since secured patent rights from the American, British, French, German and Japanese Governments.

The principle of this technique is: the material to be examined, for instance the fly's head, is pressed on a thin celluloid film which has been softened by some kind of solvent (e.g., amil-acetate); after several minutes, removing the material carefully from the celluloid-surface, and the film is brought under a common biological microscope for examination. Too strong illumination should be avoided. Thus, this method has much common with the papier-maché mould.

This method is simple enough, still it requires some skill to obtain a satisfactory result. Some examples of photomicrographs taken with this method are shown in Figures 12-14.

Of course, applications of this technique are not restricted to Drosophily. The communications for further informations on this method are to be addressed to: Dr. J. Suzuki, Imperial Sericultural Experiment Station, Tôkyô, Japan.

Hereditary Nosebleed

Hereditary multiple telangiectasis with epistaxis, known also as Rendu-Osler-Weber disease, is a syndrome in which the terminal vessels, the capillaries and the venules become dilated. On microscopic examination these dilated vessels are found to have a single layer of endothelial cells covered by a very thin epithelium. As a result of this peculiar anatomic construction, these vessels readily become dilated and bleed profusely following the slightest trauma.

These lesions appear grossly as small red or purplish spots, usually occurring on the nasal mucosa of the septum and turbinates and sometimes occurring on the oral mucosa and skin as seen in this patient. It is known that the telangiectases may occur anywhere in the skin or mucous membranes and may become manifest as hematuria, gastrointestinal bleeding, uterine bleeding or epistaxis.

This disease, unlike hemophilia, may be transmitted by either sex and may affect either the male or the female. Unlike hemophilia, the bleeding and coagulation time are normal.

The diagnosis depends on three factors: (1) a history of repeated hemorrhages, (2) a history of familial occurrence and (3) the discovery of telangiectatic lesions in the mucous membranes.

* * *

F., a man, aged 71, has had frequent nosebleeds since childhood. His father also suffered from frequent and severe epistaxis. The patient's nosebleeds became more severe at the age of 18 after a fracture of the nose. From this time until he was 43, he allowed the hemorrhages to continue until they stopped spontaneously. When he was 43 the bleeding increased in severity and frequency, so that he sought medical aid. Within a period of two years he underwent nine intranasal operations, including almost complete removal of the septum. The bleeding was not relieved. It recurred at frequent intervals, lasting from two to four hours.

Three years before admission to Vanderbilt Clinic, he received radium treatment at a nose and throat hospital in New York. During the treatment he suffered a severe epistaxis, which was controlled with difficulty and necessitated transfusion.

* * *

1. Submucous injection of quinine and urethane, 5 per cent solution, presents a simple and effective means of controlling hereditary multiple telangiectasis with epistaxis, a troublesome and dangerous disease.
2. Vasodilators such as alcohol and stimulants such as coffee should be forbidden in the treatment of these patients.
3. This condition is not amenable to surgery.

—George Hunter O'Kane, M.D., in *Jour. A. M. A.*, page 241, July 16, 1938.

THE KINDRED FILE

Its Value for Genetical Studies of Defective Individuals

WALTER E. SOUTHWICK

Research Department, Letchworth Village, Thiells, N. Y.

THE nature of the various hereditary and environmental factors that operate in the production of the mental and other deficiencies that eventually lead to the institutionalization of an individual is indicated from the numerous studies that have been made concerning such deficiencies. In some cases, such as those of mongolism,[1, 2, 3, 4] non-genetic factors probably are of prime importance, whereas, in others, such as amaurotic familial idiocy,[5] hereditary factors play the dominant role. Irrespective, however, of the nature of these causative factors, it is significant that their identification and study is completely dependent upon the availability of information concerning the nature of the condition and the extent of its occurrence among the near relatives of the affected individuals. A high familial incidence of such a condition, in general, indicates that hereditary factors predominate, and even where such an incidence is associated with some specialized environmental condition, that condition, in its turn, often depends, ultimately, upon genetic factors. On the other hand, an incidence that is essentially the same in the family group and in the general population indicates that non-hereditary factors predominate,—or at least that the group in question does not differ from the general population in the trait in question.

The need for information, therefore, concerning family relationships, and concerning the characteristics of the relatives is thus shown to be inescapable, but the collection of such data is, nevertheless, difficult, laborious, and costly, and so is frequently liable to provide the limiting factor in the study of human genetics. For these reasons, therefore, it is important that all possible measures be taken to facilitate the collection and to insure reliability in the recording of these sorts of data. This is especially true, in view of the large amounts of material involved, of the data that are collected routinely for the purpose of recording the social history of such individuals, or for other similar purposes.

Such routinely recorded data have generally been collected by the social service departments of institutions and have assumed the form, on the one hand, of brief descriptions of the fraternal, parental, when possible of the grand parental, and, occasionally when possible, of a few of the collateral relatives of the patient, and, on the other hand, of rather extended and detailed descriptions of the other aspects of the patient's preinstitutional environment. Information that is thus collected, however, is of but limited value in the determination of the relative effects of the hereditary and of the environmental factors, for no specific and definite record is made in such records, *both* of the negative and of the positive occurrences, in the relatives, of the characteristics or traits that appeared in the patient.

In order, however, to consider and record such quantitative degrees for the occurrences of a trait, it is necessary to have a prearranged and definitely established list of characteristics for such consideration. Such a list might tentatively be presented in four groups which would represent, respectively, the social, anthropo-anatomical, medico-physiological, and the neuro-psycho-psychiatric characteristics. In the social group would be included information concerning the nature of the early developmental environment, the education, occupation, domes-

tic or marital condition, and the socio-economic status. The anthropo-anatomical factors would include the racial background, color background, physical defects, sense organ defects, and abnormal features of the physical appearance. The medico-physiological would include illnesses and diseases, fecundity, and sexual abnormalities, while the neuro-psycho-psychiatric factors would include mental deficiencies, speech difficulties, special talents, special abilities, criminal tendencies, childhood delinquencies, and temperament characteristics. In order to conserve space in recording these data, the conditions which might occur in each of these four groups could be listed and each designated by means of a code number. This could be used for subsequent reference to the trait. The composition of these lists would change from time to time, and whenever new and significant developments should occur in the various fields that were represented.

Such a volume of data concerning the individuals related to the institutionalized persons might best be expressed on cards, devoting one card to each such individual. Such cards might best be the usual 5″ × 8″ cards, on which the data could readily be typed, and each card would contain, in addition to the coded or otherwise represented information concerning the characteristics, the name of the individual, together with his place and date of birth, his present address, the date it was last verified, or date and place of death, the case number of the institutionalized member of the family, and the kinship position. The kinship position of an individual could readily be represented by making symbols indicate the most direct line of relationship *from* the institutionalized person. Eight symbols with which to represent the father, mother, brother, sister, son, daughter, husband, and wife would amply meet all requirements.

The unit of filing for these cards would be the group of persons who are related to the institutionalized individual. Such a group has been termed a "kindred," and has been technically defined as a "group of individuals, each of whom is related in some way, either by consanguinity or by marriage, to every other member of the group, but in so far as can be known at any given time, to no other person." Each kindred would be identified by means of the case number of the institutionalized member, and would be filed according to a convenient order of increase in the magnitude of such numbers.

Within the kindred, the cards could be filed alphabetically, according to name, while each kindred could be located by means of another card, or kindred card, which would contain space for recording the case number, the total number of individuals in the kindred, and the characteristics of the patient. As with the other cards, this latter information would be recorded in code form or otherwise, but should be recorded in accordance with the four groups of characteristics that have already been described.

Value of a Kindred File

Of the advantages and uses of such a file, the most significant would probably concern the ever present and always difficult problem of collecting adequate and reliable data. With such a system, facilities would be established for the ready and direct recording of all information concerning the near kin of institutionalized individuals, whenever, and as rapidly as, such information should become available. Such information would have a permanent value for all subsequent genetico-environmental studies, and so would justify the utmost of care in its recording.

On the other hand, such a file, when reasonably well established, would markedly expedite further studies and analyses of human characteristics. With such a system, one could determine, by direct observation, the characteristics of any patient, could select, by means of a single and direct examination, all of the cases which show any of the listed characteristics, or could readily determine the exact extent to which any such listed trait is distributed among the near relatives of a patient.

In these ways, the genetic significance

of any characteristic could readily be determined. Traits which appear two or more times in one kindred would be more likely to have been produced by genetic factors than would those that consistently occur but once in a kindred. In many cases, more than one member of a kindred may have been admitted to an institution, and in such cases, further study in order to determine the nature of the hereditary mechanism would be well justified.

When two or more interrelated persons are admitted to the same institution, the matter of clearly expressing the kinship position of the other persons related to such patients requires some special consideration. The simple system heretofore described was selected because of its *specific* suitability for expressing the kinship relationships of persons related to institutionalized individuals. It is based upon the concept of a single fixed point, the patient, in relation to which, the positions of all of the relatives of the patient are expressed. It is essential, therefore, that, irrespective of the size of the kindred, there should never occur more than one "fixed point." When another member of the family, therefore, is admitted to the institution, it is necessary that the kinship position of the new patient be expressed in relation to the first admission. The case number of the first admission should be retained for the kindred.

Thus the small groups of interrelated persons, or "centers" within a vast existent but undescribed whole of interrelated persons, would, from time to time, fuse and become mingled into single, but proportionally larger "centers". As more such "centers" become established, and as they progressively and continually enlarge by subsequent admissions of relatives to the same institution, relationships between the relatives of *already* institutionalized individuals from two or more different "centers" would frequently be discovered. As these relationships would be described and the fusions effected, the sizes of the "centers" would be still further enlarged, and, theoretically at least, at an ever increasing rate.

Finally, due to the fact that the birth of an individual has created a kinship relationship that has eternal existence, every kinship relationship, when once it has been created, is irreversible and indestructible. For this reason, the process of discovering and recording such interrelationships would be a continually progressive one, and this permanency of kinship relationships would, to an extent, justify the expenditure of such time and energy as may be necessary for their accurate and adequate recording.

Summary

Information concerning the persons who are most closely related to institutionalized individuals is essential for any study or analysis of the hereditary and environmental causes of human deficiencies. In order to facilitate the collection and to ensure a satisfactory completeness in such information, a method for the concise and direct recording and filing on cards of such data has been devised and is described.

Such a file would provide facilities for the ready and direct recording of all information concerning the persons related to institutionalized individuals, and, as soon as it had become reasonably well established, would markedly expedite further studies and analyses of human characteristics, while the number of interconnections among the groups of such interrelated persons would show a permanent, progressive, continuous, and irreversible increase.

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Material for Demonstrating Salivary Gland Chromosomes

Interrelated demonstration material—microscopic slides, chromosome maps, lantern slides and literature—make giant chromosomes available for classroom use.

MICROSCOPIC SLIDES OF SALIVARY GLAND CHROMOSOMES

Slide of Normal Salivary Chromosomes of *D. melanogaster*, each	$2.25
Three Slides	5.00
Slide with drawing identifying all chromosomes in two figures (Female)	4.00*
Slide showing Y-chromosome (Male)	4.00
Slide showing synapsis of normal and inverted X-chromosome (loop)	4.00*
Slide showing figure of an autosomal inversion	4.00*
Slide showing synapsis of translocated and normal chromosomes	4.00*
Slide showing a small deficiency (deletion)	4.00*
Salivary chromosomes of *D. virilis*	2.25
Three Slides	5.00
Normal Somatic (ganglion) chromosomes of *D. melanogaster*	2.50
(Magnification of at least 1000 × necessary to view satisfactorily.)	
1 Slide of each of above (9 slides—6 drawings)	25.00

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Painter's cytogenetic map of the salivary chromosomes, 9½ by 18 inches, line-cut showing major chromosome details and the genes approximately located to end of 1934, mailed unfolded	$ .50
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Bridges' Revised Map of the X-Chromosome—9½" x 18", unfolded	.75
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1 copy each of Painter's, Hughes', and Bridges' two maps	2.50

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† Volume and page number of illustration in the JOURNAL OF HEREDITY.

ILLUSTRATED LITERATURE

"Symposium Reprint" on Salivary Gland Chromosomes, containing articles from the JOURNAL OF HEREDITY, 1934-1938, by Painter, Bridges, Metz, Mackensen and Hughes—four Salivary Gland Chromosome maps, 15 illustrations, 54 pages $3.00

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